Acta vet. scand. 2000, *41,* 243-247.

Mastitis in the Lactating Mink Female (*Mustela vison S.*) and the Development of "Greasy Kits"

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Clausen TN, Dietz HH: Mastitis in the lactating mink female (*Mustela vison S.*) and the development of "greasy kits". Acta vet. scand. 2000, 41, 243-247. – "Greasy kits" is the result of a multifactorial disease complex with few known definitive aetiological factors. Mastitis has been hypothesized as a triggering factor although classical clinical signs of mastitis (rubor, tumor, dolor, calor) are rarely seen in lactating Danish mink females. In this study we sacrificed 2 groups of lactating mink females with a total of 78 mammary glands at day 19-30 after giving birth. The first group had raised normal mink kits while the other group had suffered severe attacks of greasy kits. We found no clinical or histopathological evidence of mastitis but isolated streptococci and staphylococci from 2 mammary glands in females raising greasy kits. These glands showed no clinical or histological signs of inflammation attributable to bacteria and we conclude that mastitis is not necessary for the generation of greasy kits.

mink; Mustela vison S.; greasy kits; mastitis.

Introduction

During the lactation period cervical apocrine adenitis or "greasy kits" is a common disease among mink kits in Denmark. Greasy kits is probably a multifactorial disease complex with few known definitive releasing factors. Litters with many kits from young females giving birth late in the period are at greatest risk (*Olesen & Clausen* 1990). Very high proportions of fat in the feed during the lactation period may increase this problem (*Olesen & Clausen* 1992). Bacteriological examination of greasy kits shows predominantly *Staphylococcus spp.* in kits up to 2 weeks of age and *Escherichia coli* in older kits (*Rattenborg et al.* 1995). Various E. coli serotypes are prevalent but no difference in serotypes or presence of virulence factors between healthy and diseased kits was found (*Jørgensen et al.* 1996).

No clear epidemiological evidence of an infection spreading in time and space during outbreaks of greasy kits has been found (*Chriél et al.* 1997). Thus the greasy kit condition is most likely due to a secondary infection in weakened kits, rather than a primary bacterial infection. However, mild symptoms of greasy kits can be provoked by oral inoculation with *E. coli* and Staphylococci (*Henriksen,* personal communication).

Svansson (1991) found that intraperitoneal inoculation of a reovirus-like virus could cause symptoms resembling greasy kits, and *Järplid & Mejerland* (1998) found histological alterations in the intestinal villus epithelium of greasy kits resembling changes caused by rotavirus infection in neonatal calves. An "atypical" rotavirus causing diarrhoea in 2 to 6 weeks

old ferret kits was isolated by *Torres-Medina* (1987).

In mink production management problems as a contributing factor in development of this disease, have not yet been thoroughly investigated. Mastitis in the females has been hypothesized as a triggering factor (*Trautwein & Helmboldt* 1966, *Henriksen* 1988) although classical clinical signs of mastitis (rubor, tumor, dolor, calor) are rarely seen in Danish lactating mink females. This is opposed to an investigation by *Schneider & Hunter* (1993) who found, that 11 % of mink females dying in the lactation period suffered from clinical mastitis.

The present study was performed to ascertain whether there was clinical or subclinical mastitis in female mink with greasy kit litters.

Materials and methods

Animals and housing

Female, Scanbrown mink were housed at the Danish Fur Breeders Research Centre during the lactation period 1998. The females were fed a standard mink diet from the local feed kitchen during the observation period.

The animals were housed in standard mink cages (90 cm long, 30 cm wide and 45 cm high) with wooden nest boxes (30 cm × 24 cm × 20 cm) attached. The nest boxes were equipped with straw. The animals were fed once daily on top of the wire cage until the 10th May, and twice a day for the rest of the period. Through out the study all animals had free access to tap water by means of an automatic watering system.

All procedures involving handling and management of the animals were carried out according to guidelines approved by the member states of the Council of Europe for the protection of vertebrate animals used for experimental and other scientific purposes (*Anon.* 1986).

Greasy kits and clinical mastitis

All litters at the farm were inspected daily for the presence of greasy kits. Greasy kits were defined as mink kits showing a greasy, sticky exudate on the skin surface especially in the neck region as well as on the claws and tail, red and swollen perianal region, often a yellowish-white diarrhoea and invariably a moaning behaviour. Seven female mink where all the kits in the litter had severe greasy kits symptoms were chosen as well as a group of 5 female mink with normal kits. The mammary glands of the females were examined visually and by palpation for classical clinical signs of mastitis (rubor, tumor, dolor, calor).

Post mortem examinations

Seven females with sticky kits and 5 females without sticky kits were euthanised with sodium pentobarbital (25 mg kg^{-1} body weight, intraperitoneally). The skin of the mammary glands was disinfected by slight, superficial burning with a gas flame. Each gland was incised with a sterile knife and samples were taken for bacteriological and histological examination, to evaluate, if there were any signs of subclinical mastitis, defined as specific bacterial growth and inflammatory lesions in the gland.

The glands were identified individually as R (right row) or L (left row) and numbered consecutively in each individual with no. 1 being the most cranial gland.

Bacteriological examination of specimens

Immediately after euthanisation samples from the incised mammary gland were taken with a sterile Miniplast Ein-shemer (Quadloop). The samples were processed immediately by streaking onto Blood Agar base no. 2 (OXOID, CM271) and incubated aerobically at 37°C overnight. Colonies surrounded by a clear halo were defined as exibiting haemolytic activity.

Colonies exibiting streptococci- and staphylococci-like growth were verified as streptococci and staphylococci by the API20 STREP system (BioMerieux) and the API STAPH system (BioMerieux) respectively.

Histology

All the mammary glands, including the overlying skin, from each of the females were dissected from the abdominal wall, separated from the neighbouring glands and fixed in 10% neutral buffered formalin in separate jars. Subsequently the mammary glands were dissected and a slide with the central (large) portion including the nipple was embedded in Paraplast[R] according to routine procedures. Four μm sections were stained with haematoxylin and eosin. All sections were evaluated in a Leitz, Diaplan microscope at 25, 100 and 400 × magnification.

Results

There were no macroscopic signs of mastitis in any of the females. The bacteriological examination showed sparse bacterial growth in 2 out of 78 glands. The bacteria were characterized as non-haemolytic streptococci and *Staphylococcus aureus* respectively. The histological examination showed no signs of inflammation in the mammary tissue proper in these 2 glands. The rest of the glands were sterile.

The histological examination furthermore revealed that all nipples had a normal hyperkeratotic and slightly irregular epidermal hyperplasia. The hair follicles contained numerous hair shafts. The perifollicular area and the hair adnexal structures were highly oedematous and contained varying amounts of a diffusely dispersed, mixed cellular infiltrate dominated by eosinophilic granulocytes and a few lymphocytes and plasma cells.

Histologically the mammary glandular tissue exhibited varying stages of secretory activity and some lobules showed degenerative changes (involution). A mild inflammation in R3 in one mink, was characterized by one lobule undergoing atrophy and showing signs of chronic inflammation.

Staphylococci isolated from R4 in one mink did not give rise to inflammation but the glands exhibited congestion and focal bleeding.

Discussion

Greasy kits is a multifactorial disease with few known eliciting factors. Greasy kits is a problem known in all mink producing countries in the Northern hemisphere (*Aldén & Mejerland* 1997, *Sluggin* (pers. comm.)). *Hunter & Schneider* (1996) postulated that greasy kits or adenitis of the neonatal cervical gland occurs frequently in mutation colour variants of mink. Our experience is that all colour types are affected, but the most serious outbreaks is usually seen in black and blue type mink.

An array of predisposing factors have been evaluated in Denmark (*Uttenthal et al.* (1999), *Elnif & Engaard* (1997), *Chriél et al.* (1997), *Chriél* (1997), *Rattenborg et al.* (1995), *Clausen* (1993)). It has turned out to be very difficult to perform prospective as well as experimental studies of greasy kits as the morbidity rate varies annually from 0 to >20 % of the litters in a farm.

Although an infectious aetiology for greasy kits is obvious, similar to that of diarrhoea in newborn calves and pigs, we have not succeeded experimentally in showing a causal impact of Danish bacterial isolates from greasy kits.

Restriction in feed intake in February followed by ad libitum feeding in March is often applied to female mink prior to breeding (*Atkinson* 1996). Some mink farmers, however seem to reduce the energy intake too much and epidemiological examination of large data sets have

shown that a low energy intake in the breeding period predisposes for greasy kits in the following whelping period (*Chriél* 1997).

Mastitis is considered as an inflammatory disease rather than an infectious disease (*Jubb & Kennedy* 1993) and the pathological alterations with mastitis caused by bacteria are characterized by interstitial oedema, extensive migration of neutrophils into the interlobular tissue and secretory acini. Fibrosis and involution takes place very rapidly and macrophages and fibroblasts increase in number (*Jubb & Kennedy* 1993).

According to *Löliger* (1970) the clinical signs may include swelling, firmness, abscessation of affected glands and necrotizing mastitis. Histologically the teat canal and alveoli may contain exudate and the interstitia are oedematous with small clusters of neutrophils.

We saw no such signs in this study. The involution described is considered a normal phenomenon before weaning and the dermal alterations are attributed to intensive suckling and padling behaviour of the mink kits. Furthermore the epidermis and dermis were histologically similar among normal females and females with greasy kits.

In conclusion mastitis does not appear to be a necessary cause for "greasy kits".

Acknowledgement

This study was supported by the Danish Fur Breeders Association Research Foundation.

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Sammendrag

Mastitis hos lakterende mink tæver (Mustela vision S) og udvikling af „fedtede hvalpe"

For at afprøve påstanden om, at det multifaktorielle syndrom "fedtede hvalpe" hos mink har sammenhæng med forekomst af mastitis hos minktæven, blev 2 grupper af minktæver repræsenterende ialt 78 mælkekirtler aflivet på 19.-30. dagen i laktationsperioden. I den ene gruppe var tævernes hvalpe fedtede, medens den anden gruppe tæver opfostrede normale hvalpe. Studiet viste ikke kliniske eller histopatologiske tegn på mastitis i nogen af grupperne, medens der fra 2 kirtler hos tæver med fedtede hvalpe isoleredes henholdvis stafylokokker og streptokokker. Det konkluderes, at mastitis hos minktæver ikke er en nødvendig faktor for udvikling af syndromet fedtede hvalpe hos mink.

(Received April 14, 2000, accepted April 14, 2000).

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